WEST Search History

DATE: Friday, July 12, 2002

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,DWPI; PLUR=YES; OP=ADJ			
L4	cardiac near3 enhancer	15	L4
L3	Csx near3 enhancer	0	L3
L2	Csx near3 enhancer	0	L2
L1	Csx near3 promoter	0	L1

END OF SEARCH HISTORY

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 NEWS 4 Feb 01 DKILIT now produced by FIZ Karlsruhe and has a new update
          frequency
 NEWS 5 Feb 19 Access via Tymnet and SprintNet Eliminated Effective 3/31/02
 NEWS 6 Mar 08 Gene Names now available in BIOSIS
 NEWS 7 Mar 22 TOXLIT no longer available
 NEWS 8 Mar 22 TRCTHERMO no longer available
 NEWS 9 Mar 28 US Provisional Priorities searched with P in CA/CAplus
          and USPATFULL
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 NEWS 11 Apr 02 PAPERCHEM no longer available on STN.  Use
PAPERCHEM2 instead
 NEWS 12 Apr 08 "Ask CAS" for self-help around the clock
 NEWS 13 Apr 09 BEILSTEIN Reload and Implementation of a New Subject Area
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IFIUDB
 NEWS 16 Apr 22 Records from IP.com available in CAPLUS, HCAPLUS, and
ZCAPLUS
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 NEWS 18 Apr 22 Federal Research in Progress (FEDRIP) now available
 NEWS 19 Jun 03 New e-mail delivery for search results now available
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 NEWS 21 Jun 10 PCTFULL has been reloaded

 NEWS EXPRESS  February 1 CURRENT WINDOWS VERSION IS V6.0d,
          CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),
          AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
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L5        641 MKS

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L7          0 L4 AND L6

=> s l4 and l6
L8          0 L4 AND L6

=> s nkx
L9        495 NKX

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L10      1031 L4 OR L9

=> s cardiac or heart
L11   1459500 CARDIAC OR HEART

=> s l11 and l10
L12       347 L11 AND L10

=> s l12 and enhancer
L13        30 L12 AND ENHANCER

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PROCESSING COMPLETED FOR L13
L14        21 DUP REM L13 (9 DUPLICATES REMOVED)

=> d bib abs 1-
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L14  ANSWER 1 OF 21  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS
INC
AN   2002:298130  BIOSIS
DN   PREV200200298130
TI   ***Cardiac*** -specific activity of an Nkx2-5  ***enhancer***
     requires an evolutionarily conserved Smad binding site.
AU   Lien, Ching-Ling; McAnally, John; Richardson, James A.; Olson, Eric N. (1)
CS   (1) Department of Molecular Biology, University of Texas Southwestern
     Medical Center at Dallas, 6000 Harry Hines Boulevard, Dallas, TX, 75390
     eolson@hamon.swmed.edu USA
SO   Developmental Biology, (April 15, 2002) Vol. 244, No. 2, pp. 257-266.
     http://www.academicpress.com/db/ print.
     ISSN: 0012-1606.
DT   Article
LA   English
AB   ***Heart***  formation in vertebrates and fruit flies requires
     signaling by bone morphogenetic proteins (BMPs) to cardiogenic mesodermal
     precursor cells. The vertebrate homeobox gene Nkx2-5 and its Drosophila
     ortholog, tinman, are the earliest known markers for the  ***cardiac***
     lineage. Transcriptional activation of tinman expression in the
      ***cardiac***  lineage is dependent on a mesoderm-specific
      ***enhancer***  that binds Smad proteins, which activate transcription in
     response to BMP signaling, and Tinman, which maintains its own expression
     through an autoregulatory loop. Here, we show that an evolutionarily
     conserved,  ***cardiac*** -specific  ***enhancer***  of the mouse
     Nkx2-5 gene contains multiple Smad binding sites, as well as a binding
     site for Nkx2-5. A single Smad site is required for  ***enhancer***
     activity at early and late stages of  ***heart***  development in vivo,
     whereas the Nkx2-5 site is not required for  ***enhancer***  activity.
     These findings demonstrate that Nkx2-5, like tinman, is a direct target
     for transcriptional activation by Smad proteins, however, the independence
     of this Nkx2-5  ***enhancer***  of Nkx2-5 binding suggests a fundamental
     difference in the transcriptional circuitry for activation of Nkx2-5 and
     tinman expression during cardiogenesis in vertebrates and fruit flies.

L14  ANSWER 2 OF 21  CAPLUS  COPYRIGHT 2002 ACS
AN   2001:525880  CAPLUS
DN   135:127154
TI   Sequences of  ***cardiac*** -cell specific  ***enhancer***  elements
     from human and mouse.  ***Csx***  /Nkx2.5 gene regulatory regions and
     therapeutic uses thereof in inducing the differentiation of stem cells as
     cardiomyocytes.
IN   Lee, Ike W.; Izumo, Seigo
PA   Beth Israel Deaconess Medical Center, USA
SO   PCT Int. Appl., 66 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
```

PATENT NO KIND DATE APPLICATION NO DATE

PI WO 2001051006 A2 20010719 WO 2001-US1511 20010116
WO 2001051006 A3 20011220
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
AU 2001034470 A5 20010724 AU 2001-34470 20010116
US 2002022259 A1 20020221 US 2001-761466 20010116
PRAI US 2000-176419P P 20000114
WO 2001-US1511 W 20010116

AB The invention provides sequences of ***cardiac*** cell-specific ***enhancer*** elements derived from human and mouse ***Csx*** /Nkx2 5 regulatory regions. These ***enhancer*** elements are useful, for example, for (i) regulating gene expression in ***cardiac*** cells, (ii) inducing stem cells (e g , embryonic stem cells or bone marrow stem cells) to differentiate as cardiomyocytes, and (iii) identifying factors that induce the differentiation of stem cells as cardiomyocytes

L14 ANSWER 3 OF 21 CAPLUS COPYRIGHT 2002 ACS
AN 2001 489582 CAPLUS
DN 135 104695
TI Cells capable of differentiating into ***heart*** muscle cells
IN Umezawa, Akihiro, Hata, Jun-ichi, Fukuda, Keiichi, Ogawa, Satoshi, Sakurada, Kazuhiro, Gojo, Satoshi, Yamada, Yoji
PA Kyowa Hakko Kogyo Co , Ltd , Japan
SO PCT Int Appl , 183 pp
CODEN PIXXD2
DT Patent
LA Japanese
FAN CNT 3

PATENT NO KIND DATE APPLICATION NO DATE

PI WO 2001048151 A1 20010705 WO 2000-JP9323 20001227
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
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WO 2001048149 A1 20010705 WO 2000-JP1148 20000228
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
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WO 2001048150 A1 20010705 WO 2000-JP7741 20001102
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
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PRAI JP 1999-372826 A 19991228
WO 2000-JP1148 W 20000228
WO 2000-JP7741 W 20001102

AB Methods are described for isolating, purifying, culturing and differentiation-inducing cells capable of differentiating into ***heart*** muscle cells. A method is described for proliferating cells capable of differentiating into ***heart*** muscle cells by using various cytokines, transcription factors, or else. A method is described for regulating the differentiation of cells into ***heart*** muscle cells by using various cytokines, transcription factors, or else. A method is described for obtaining a surface antigen specific to cells capable of differentiating into ***heart*** muscle cells. A method is described for obtaining a gene encoding this surface antigen. A method is described for obtaining an antibody specific to the surface antigen. A method is described for obtaining a protein and a gene participating in the proliferation and differentiation into ***heart*** muscle cells of cells capable of differentiating into ***heart*** muscle cells. Drugs for various ***heart*** diseases using cells capable of differentiating into ***heart*** muscle cells are described. A method is described for inducing the differentiation of various cells and tissues such as nerve cells, liver cells, fat cells, skeletal muscle cells, vascular endothelial cells and osteoblasts by using cells capable of differentiating into ***heart*** muscle cells

RE CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 4 OF 21 CAPLUS COPYRIGHT 2002 ACS
AN 2001 489581 CAPLUS
DN 135 104694
TI Cells capable of differentiating into ***heart*** muscle cells
IN Umezawa, Akihiro, Hata, Jun-ichi, Fukuda, Keiichi, Ogawa, Satoshi, Sakurada, Kazuhiro, Gojo, Satoshi, Yamada, Yoji
PA Kyowa Hakko Kogyo Co , Ltd , Japan
SO PCT Int Appl , 187 pp
CODEN PIXXD2
DT Patent
LA Japanese
FAN CNT 3

PATENT NO KIND DATE APPLICATION NO DATE

PI WO 2001048150 A1 20010705 WO 2000-JP7741 20001102
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
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WO 2001048149 A1 20010705 WO 2000-JP1148 20000228
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WO 2001048151 A1 20010705 WO 2000-JP9323 20001227
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PRAI JP 1999-372826 A 19991228
WO 2000-JP1148 W 20000228
WO 2000-JP7741 W 20001102

AB Methods are described for isolating, purifying, culturing and differentiation-inducing cells capable of differentiating into ***heart*** muscle cells. A method is described for proliferating cells capable of differentiating into ***heart*** muscle cells by using various cytokines, transcription factors, or else. A method is described for regulating the differentiation of cells into ***heart*** muscle cells by using various cytokines, transcription factors, or else. A method is described for obtaining a surface antigen specific to cells capable of differentiating into ***heart*** muscle cells. A method is described for obtaining a gene encoding this surface antigen. A method is described for obtaining an antibody specific to the surface antigen. A method is described for obtaining a protein and a gene participating in the proliferation and differentiation into ***heart*** muscle cells of cells capable of differentiating into ***heart*** muscle cells. Drugs for various ***heart*** diseases using cells capable of differentiating into ***heart*** muscle cells are described. A method is described for inducing the differentiation of various cells and tissues such as nerve cells, liver cells, fat cells, skeletal muscle cells, vascular endothelial cells and osteoblasts by using cells capable of differentiating into ***heart*** muscle cells

RE CNT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 5 OF 21 CAPLUS COPYRIGHT 2002 ACS
AN 2001 489580 CAPLUS
DN 135 89527
TI Adult bone marrow-origin cell capable of differentiating into ***heart*** muscle cell
IN Umezawa, Akihiro, Hata, Junichi, Fukuda, Keiichi, Ogawa, Satoshi, Sakurada, Kazuhiro
PA Kyowa Hakko Kogyo Co , Ltd , Japan
SO PCT Int Appl , 158 pp
CODEN PIXXD2
DT Patent
LA Japanese
FAN CNT 3

PATENT NO KIND DATE APPLICATION NO DATE

PI WO 2001048149 A1 20010705 WO 2000-JP1148 20000228
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WO 2001048150 A1 20010705 WO 2000-JP7741 20001102
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WO 2001048151 A1 20010705 WO 2000-JP9323 20001227
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
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PRAI JP 1999-372826 A 19991228
WO 2000-JP1148 W 20000228
WO 2000-JP7741 W 20001102
AB Methods are described for isolating, purifying, culturing and differentiation-inducing bone marrow cells capable of differentiating into ***heart*** muscle cells. A method is described for proliferating bone marrow cells capable of differentiating into ***heart*** muscle cells by using various cytokines, transcription factors, or else. A method is described for regulating the differentiation of bone marrow cells into ***heart*** muscle cells by using various cytokines, transcription factors, or else. A method is described for obtaining a surface antigen specific to bone marrow cells capable of differentiating into ***heart*** muscle cells. A method is described for obtaining a gene encoding this surface antigen. A method is described for obtaining an antibody specific to the surface antigen. A method is described for obtaining a protein and a gene participating in the proliferation and differentiation into ***heart*** muscle cells of bone marrow cells capable of differentiating into ***heart*** muscle cells. Drugs for various ***heart*** diseases using bone marrow cells capable of differentiating into ***heart*** muscle cells are described
RE CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 6 OF 21 CAPLUS COPYRIGHT 2002 ACS
AN 2001:354388 CAPLUS
DN 135:105661
TI Growth and gene expression profile analyses of endometrial cancer cells expressing exogenous PTEN
AU Matsushima-Nishiu, Mieko, Unoki, Motoko, Ono, Kenji, Tsunoda, Tatsuhiko, Minaguchi, Takeo, Kuramoto, Hiroyuki, Nishida, Masato, Satoh, Toyomi, Tanaka, Toshihiro, Nakamura, Yusuke
CS Laboratories of Molecular Medicine, Human Genome Center, Institute of Medical Science, The University of Tokyo, Tokyo, 108-8639, Japan
SO Cancer Research (2001), 61(9), 3741-3749
CODEN: CNREA8, ISSN: 0008-5472
PB American Association for Cancer Research
DT Journal
LA English
AB The PTEN tumor suppressor gene encodes a multifunctional phosphatase that plays an important role in inhibiting the phosphatidylinositol-3-kinase pathway and downstream functions that include activation of Akt/protein kinase B, cell survival, and cell proliferation. Enforced expression of PTEN in various cancer cell lines decreases cell proliferation through arrest of the cell cycle, accompanied in some cases by induction of apoptosis. We used cDNA microarrays contg. 4009 cDNAs to examine changes in gene-expression profiles when exogenous PTEN was induced in PTEN-defective cells. The microarrays and subsequent semiquant. reverse transcription-PCR anal. revealed transcriptional stimulation of 99 genes and repression of 72 genes. Some of the differentially expressed genes already had been implicated in cell proliferation, differentiation, apoptosis, or cell cycle control, e.g., overexpression of PTEN-induced transactivation of cyclin-dependent inhibitor 1B (p27Kip1) and 2B (p15INK4B), members of the TNF receptor family, tumor necrosis factor-assocd. genes, and members of the Notch-signaling and Mad families. To our knowledge this is the first report of transactivation of those genes by PTEN. The genes differentially expressed in our expts. also included many whose correlation with cancer development had not been recognized before. Our data should contribute to a greater understanding of the broad spectrum of ways in which PTEN affects intracellular signaling pathways. Anal. of expression profiles with microarrays appears to be a powerful approach for identifying anticancer genes and/or disease-specific targets for cancer therapy.
RE CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 7 OF 21 CAPLUS COPYRIGHT 2002 ACS
AN 2001:549785 CAPLUS
DN 136:132747
TI Transplanted adult bone marrow cells repair myocardial infarcts in mice
AU Orlic, Donald, Kajstura, Jan, Chimenti, Stefano, Bodine, David M., Leri, Annarosa, Anversa, Piero
CS Genetics and Molecular Biology Branch, National Human Genome Research Institute, NIH, Bethesda, MD, 20892, USA
SO Annals of the New York Academy of Sciences (2001), 938(Hematopoietic Stem Cells 2000), 221-230
CODEN: ANYAA9, ISSN: 0077-8923
PB New York Academy of Sciences
DT Journal
LA English
AB Occlusion of the anterior descending left coronary artery leads to ischemia, infarction, and loss of function in the left ventricle. We have studied the repair of infarcted myocardium in mice using highly enriched stem/progenitor cells from adult bone marrow. The left coronary artery was ligated and 5 h later Lin- c-kit+ bone marrow cells obtained from transgenic male mice expressing enhanced green fluorescent protein (EGFP) were injected into the healthy myocardium adjacent to the site of the infarct. After 9 days the damaged hearts were examd. for regenerating myocardium. A band of new myocardium was obsd. in 12 surviving mice. The developing myocytes were small and resembled fetal and neonatal myocytes. They were pos. for EGFP, Y chromosome, and several myocyte-specific proteins including ***cardiac*** myosin and the transcription factors GATA-4, MEF2, and ***Csx*** /Nkx2.5. The cells were also pos. for connexin 43, a gap junction/intercalated disk component indicating the onset of intercellular communication. Myocyte proliferation was demonstrated by incorporation of BrdU into the DNA of dividing cells and by the presence of the cell cycle-assocd. protein Ki67 in their nuclei. Neo-vascularization was also obsd. in regenerating myocardium. Endothelial and smooth muscle cells in developing capillaries and small arterioles were EGFP-pos. These cells were pos. for Factor VIII and alpha smooth muscle actin, resp. No myocardial regeneration was obsd. in damaged hearts transplanted with Lin- c-kit- bone marrow cells, which lack bone marrow-regenerating activity. Functional competence of the repaired left ventricle was improved for several hemodynamic parameters. These in vivo findings demonstrate the capacity of highly enriched Lin- c-kit+ adult bone marrow cells to acutely regenerate functional myocardium within an infarcted region.
RE CNT 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 8 OF 21 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 2000424761 EMBASE
TI Genomic organization and mapping of mouse CDV (carnitine deficiency-associated gene expressed in ventricle)-1 and its related CDV-1R gene
AU Higashi M., Kobayashi K., Iijima M., Wakana S., Horiuchi M., Yasuda T., Yoshida G., Kanmura Y., Saheki T.
CS T. Saheki, Department of Biochemistry, Faculty of Medicine, Kagoshima University, Sakuragaoka, Kagoshima 890-8520, Japan
takesah@med2.kufm.kagoshima-u.ac.jp
SO Mammalian Genome, (2000) 11/12 (1053-1057)
Refs: 28
ISSN: 0938-8990 CODEN: MAMGEC
CY United States
DT Journal, Article
FS 022 Human Genetics
029 Clinical Biochemistry
LA English
SL English
AB We have previously reported that CDV (carnitine deficiency-associated gene expressed in ventricle)-1 was a down-regulated gene in the hypertrophied ventricle of carnitine-deficient juvenile visceral steatosis mice and that the related gene (CDV-1R) showed no tissue specificity and no sensitivity to carnitine deficiency. In the present paper, the CDV-1/1R gene was isolated from a mouse genomic BAC library, and the genomic structure was characterized. We found that the CDV-1/1R gene consisted of at least 19 exons and encompassed approximately 48 kb. The splice sites conformed to the GT-AG rule, and the CDV-1R mRNA containing 19 exons was processed. CDV-1 mRNA containing 5 exons was constructed from the 3' half of CDV-1R. The first exon of CDV-1 consisted of the 3' side (116 bp) of intron 14 and exon 15 (87 bp) of CDV-1R. The presumed promoter sequence for CDV-1 located in the intron 14 of CDV-1R contained the common TATA box and consensus binding sites for various transcription factors (***Nkx*** -2.5, Sp1, C/EBP, SRF, YY1, and CREB), which seem to play roles in the ***heart*** -specific expression and carnitine deficiency-associated suppression of CDV-1. In the upstream region of the CDV-1 promoter, we found two VNTRs: 13 repeats of GATA1, and 16 copies of STRE involved in yeast stress response. The CDV-1/1R gene was located close to D5MIT68 on mouse Chromosome (Chr) 5, corresponding to human Chr 12q24. All these data revealed that two mRNA species, CDV-1 and CDV-1R, are expressed tissue-specifically by using promoters peculiar to each transcript in a single gene.

L14 ANSWER 9 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE
1

AN 2000:114835 BIOSIS
DN PREV200000114835
TI An ***Nkx*** -dependent ***enhancer*** regulates cGATA-6 gene expression during early stages of ***heart*** development
AU Davis, Dorene L.; Wessels, Andy; Burch, John B. E. (1)
CS (1) Fox Chase Cancer Center, Philadelphia, PA, 19111 USA
SO Developmental Biology, (Jan. 15, 2000) Vol. 217, No. 2, pp. 310-322. ISSN: 0012-1606.
DT Article
LA English
SL English
AB The evolutionarily conserved GATA-6 transcription factor is an early and persistent marker of ***heart*** development in diverse vertebrate species. We previously found evidence for a functionally conserved ***heart*** -specific ***enhancer*** upstream of the chicken GATA-6 (cGATA-6) gene and in the present study we used transgenic mouse assays to further characterize this regulatory module. We show that this ***enhancer*** is activated in committed precursor cells within the ***cardiac*** crescent and that it remains active in essentially all cardiogenic cells through the linear ***heart*** stage. Although this ***enhancer*** can account for cGATA-6 gene expression early in the cardiogenic program, it is not able to maintain expression throughout the ***heart*** later in development. In particular, the ***enhancer*** is sequentially downregulated along the posterior to anterior axis, with activity becoming confined to outflow tract myocardium. Enhancers with similar properties have been shown to regulate the early ***heart*** -restricted expression of the mouse Nkx2.5 transcription factor gene. Whereas these Nkx2.5 enhancers are GATA-dependent, we show that the cGATA-6 ***enhancer*** is ***Nkx*** -dependent. We speculate that these enhancers are silenced to allow GATA-6 and Nkx2.5 gene expression to be governed by region-specific enhancers in the multichambered ***heart***

L14 ANSWER 10 OF 21 CAPLUS COPYRIGHT 2002 ACS
AN 1999:795994 CAPLUS
DN 132:31744
TI Gene probes used for genetic profiling in healthcare screening and planning
IN Roberts, Gareth Wyn
PA Genostic Pharma Ltd., UK
SO PCT Int. Appl., 745 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9964627	A2	19991216	WO 1999-GB1780	19990604
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				

PRAI GB 1998-12099 A 19980606
GB 1998-13291 A 19980620
GB 1998-13611 A 19980624
GB 1998-13835 A 19980627
GB 1998-14110 A 19980701
GB 1998-14580 A 19980707
GB 1998-15438 A 19980716
GB 1998-15574 A 19980718
GB 1998-15576 A 19980718
GB 1998-16085 A 19980724
GB 1998-16086 A 19980724
GB 1998-16921 A 19980805
GB 1998-17097 A 19980807
GB 1998-17200 A 19980808
GB 1998-17632 A 19980814
GB 1998-17943 A 19980819

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response. In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the no. of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide crit. clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies which comprises of the identification of the core group of genes and their sequence variants required to provide a broad base of clin. prognostic information - "genostics". The "Genostic RTM" profiling of patients and persons will radically enhance the ability of clinicians, healthcare professionals and other parties to plan and manage healthcare provision and the targeting of appropriate healthcare resources to those deemed most in need. The use of this invention could also lead to a host of new applications for such profiling technologies, such as identification of persons with particular work or environment related risk, selection of applicants for employment, training or specific opportunities or for the enhancing of the planning and organization of health services, education services and social services.

L14 ANSWER 11 OF 21 CAPLUS COPYRIGHT 2002 ACS
AN 1999:795993 CAPLUS
DN 132:31743
TI Gene probes used for genetic profiling in healthcare screening and planning
IN Roberts, Gareth Wyn
PA Genostic Pharma Limited, UK
SO PCT Int. Appl., 149 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9964626	A2	19991216	WO 1999-GB1779	19990604
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9941586	A1	19991230	AU 1999-41586	19990604
AU 9941587	A1	19991230	AU 1999-41587	19990604
GB 2339200	A1	20000119	GB 1999-12914	19990604
GB 2339200	B2	20010912		
EP 1084273	A1	20010321	EP 1999-925207	19990604
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRAI GB 1998-12098 A 19980606
GB 1998-28289 A 19981223
GB 1998-16086 A 19980724
GB 1998-16921 A 19980805
GB 1998-17097 A 19980807
GB 1998-17200 A 19980808
GB 1998-17632 A 19980814
GB 1998-17943 A 19980819
WO 1999-GB1779 W 19990604

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response. In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the no. of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide crit. clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies.

L14 ANSWER 12 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC. DUPLICATE 2
AN 1999:249774 BIOSIS
DN PREV199900249774
TI Complex modular cis-acting elements regulate expression of the ***cardiac*** specifying homeobox gene ***Csx*** /Nkx2.5.
AU Tanaka, Makoto; Burns, Wechsler, Stephanie (1); Lee, Ike W.; Yamasaki, Naohito; Lawitts, Joel A.; Izumo, Seigo (1)
CS (1) Cardiovascular Division, Department of Medicine, Harvard Medical School, 330 Brookline Avenue, Boston, MA, 02215 USA
SO Development (Cambridge), (April, 1999) Vol. 126, No. 7, pp. 1439-1450. ISSN: 0950-1991.
DT Article
LA English
SL English
AB The murine homeobox gene ***Csx*** /Nkx2.5 is an evolutionarily highly conserved gene related to the Drosophila tinman gene, which specifies ***cardiac*** and visceral mesoderm. Since ***Csx*** /Nkx2.5 plays an essential role in ***heart*** development, studying its regulation is essential for the better understanding of molecular mechanisms of cardiogenesis and the pathogenesis of congenital ***heart*** disease in humans. In this study, we characterized the murine ***Csx*** /Nkx2.5 gene and identified two novel untranslated exons, 1a, and 1b, resulting in three different ***Csx*** /Nkx2.5 transcripts. To examine the tissue-specific transcriptional regulation in vivo, we analyzed a total of

23 kb of ***Csx*** /Nkx2.5 upstream and downstream sequences by generating transgenic embryos carrying lacZ reporter constructs containing various lengths of flanking sequence. With 14 kb of 5' flanking sequence, lacZ expression was observed in the ***cardiac*** crescent at E7.5, and in the outflow tract, the interatrial groove, the atrioventricular canal and right and left ventricles, as well as in pharyngeal floor, thyroid primordia, and stomach at E10.5. In adult animals, lacZ expression of the transgene was limited to the atrioventricular junction and the subendocardium of the ventricular septum. Reducing the size of flanking sequence to 3.3 kb of intron 2 restricted lacZ expression to the outflow tract and the basal part of the right ventricle in E10.5 embryos. In contrast, the addition of 6 kb of 3' flanking sequence caused strong expression of the reporter gene in the entire right ventricle. Interestingly, ***Csx*** /Nkx2.5 seems to be negatively regulated by its own gene product, because when lacZ was "knocked-in" to replace the entire coding exons, lacZ expression was much higher in the ***heart*** of homozygous embryos than that in the heterozygote. These results indicate that the transcriptional regulatory elements of ***Csx*** / ***Nkx*** 2.5 seems unexpectedly highly modular, and is temporally regulated in a dynamic manner by different ***enhancer*** regions. Since ***Csx*** /Nkx2.5-like genes are expressed in all species having a ***heart*** , their complex modular organization with multiple enhancers probably reflects progressive addition of regulatory elements during the evolution from a simple ***heart*** tube to a complex four-chambered organ.

L14 ANSWER 13 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:113290 BIOSIS
DN PREV199900113290
TI Control of early ***cardiac*** -specific transcription of ***Nkx*** -2-5 by a GATA-dependent ***enhancer***.
AU Lien, Ching-Ling; Wu, Chuanzhen; Mercer, Brian; Webb, Robert; Richardson, James A.; Olson, Eric N. (1)
CS (1) Dep. Mol. Biol. Oncol., Univ. Tex. Southwestern Med. Cent. Dallas, 6000 Harry Hines Blvd., Dallas, TX 75235-9148 USA
SO Development (Cambridge), (Jan., 1999) Vol. 126, No. 1, pp. 75-84. ISSN: 0950-1991.
DT Article
LA English
AB The homeobox gene Nkx2-5 is the earliest known marker of the ***cardiac*** lineage in vertebrate embryos. Nkx2-5 expression is first detected in mesodermal cells specified to form ***heart*** at embryonic day 7.5 in the mouse and expression is maintained throughout the developing and adult ***heart***. In addition to the ***heart*** , Nkx2-5 is transiently expressed in the developing pharynx, thyroid and stomach. To investigate the mechanisms that initiate ***cardiac*** transcription during embryogenesis, we analyzed the Nkx25 upstream region for regulatory elements sufficient to direct expression of a lacZ transgene in the developing ***heart*** of transgenic mice. We describe a ***cardiac*** ***enhancer*** , located about 9 kilobases upstream of the Nkx2-5 gene, that fully recapitulates the expression pattern of the endogenous gene in cardiogenic precursor cells from the onset of ***cardiac*** lineage specification and throughout the linear and looping ***heart*** tube. Thereafter, as the atrial and ventricular chambers become demarcated, ***enhancer*** activity becomes restricted to the developing right ventricle. Transcription of Nkx2-5 in pharynx, thyroid and stomach is controlled by regulatory elements separable from the ***cardiac*** ***enhancer*** . This distal ***cardiac*** ***enhancer*** contains a high-affinity binding site for the ***cardiac*** -restricted zinc finger transcription factor GATA4 that is essential for transcriptional activity. These results reveal a novel GATA-dependent mechanism for activation of Nkx2-5 transcription in the developing ***heart*** and indicate that regulation of Nkx2-5 is controlled in a modular manner, with multiple regulatory regions responding to distinct transcriptional networks in different compartments of the developing ***heart***

L14 ANSWER 14 OF 21 CAPLUS COPYRIGHT 2002 ACS
AN 1999:25193 CAPLUS
DN 130:180436
TI Myocyte ***enhancer*** factor 2C and Nkx2-5 up-regulate each other's expression and initiate cardiomyogenesis in P19 cells
AU Skerjanc, Ilona S.; Petropoulos, Helen; Ridgeway, Alan G.; Wilton, Sharon
CS Department of Biochemistry, University of Western Ontario, London, ON, N6A 5C1, Can.
SO Journal of Biological Chemistry (1998), 273(52), 34904-34910. CODEN: JBCHA3; ISSN: 0021-9258.
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB The Nkx2-5 homeodomain protein plays a key role in cardiomyogenesis. Ectopic expression in frog and zebrafish embryos results in an enlarged myocardium; however, expression of Nkx2-5 in fibroblasts was not able to trigger the development of beating ***cardiac*** muscle. In order to examine the ability of Nkx2-5 to modulate endogenous ***cardiac*** specific gene expression in cells undergoing early stages of differentiation, P19 cell lines overexpressing Nkx2-5 were differentiated in the absence of Me2SO. Nkx2-5 expression induced cardiomyogenesis in these cultures aggregated without Me2SO. During differentiation into ***cardiac*** muscle, Nkx2-5 expression resulted in the activation of myocyte ***enhancer*** factor 2C (MEF2C), but not MEF2A, -B, or -D. In order to compare the abilities of Nkx2-5 and MEF2C to induce cellular differentiation, P19 cells overexpressing MEF2C were aggregated in the absence of Me2SO. Similar to Nkx2-5, MEF2C expression initiated cardiomyogenesis, resulting in the up-regulation of Brachyury T, bone morphogenetic protein-4, Nkx2-5, GATA-4, ***cardiac*** alpha -actin, and myosin heavy chain expression. These findings indicate the presence of a pos. regulatory network between Nkx2-5 and MEF2C and show that both factors can direct early stages of cell differentiation into a cardiomyogenic pathway.
RE CNT 76 THERE ARE 76 CITED REFERENCES AVAILABLE FOR THIS RECORD.
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 15 OF 21 CAPLUS COPYRIGHT 2002 ACS
AN 1999:11256 CAPLUS
DN 130:194833
TI A GATA-dependent ***nkx*** -2.5 regulatory element activates early ***cardiac*** gene expression in transgenic mice
AU Searcy, Robin D.; Vincent, Eric B.; Liberatore, Christine M.; Yutzey, Katherine E.
CS Division of Molecular Cardiovascular Biology, The Children's Hospital Research Foundation, Cincinnati, OH, 45229, USA
SO Development (Cambridge, United Kingdom) (1998), 125(22), 4461-4470. CODEN: DEVPED; ISSN: 0950-1991.
PB Company of Biologists Ltd
DT Journal
LA English
AB ***Nkx*** -2.5 is one of the first genes expressed in the developing ***heart*** of early stage vertebrate embryos. ***Cardiac*** expression of ***nkx*** -2.5 is maintained throughout development and ***nkx*** -2.5 also is expressed in the developing pharyngeal arches, spleen, thyroid and tongue. Genomic sequences flanking the mouse ***nkx*** -2.5 gene were analyzed for early developmental regulatory activity in transgenic mice. Approx. 3 kb of 5' flanking sequence is sufficient to activate gene expression in the ***cardiac*** crescent as early as E7.25 and in limited regions of the developing ***heart*** at later stages. Expression also was detected in the developing spleen anlage at least 24 h before the earliest reported spleen marker and in the pharyngeal pouches and their derivs. including the thyroid. The obsd. expression pattern from the -3 kb construct represents a subset of the endogenous ***nkx*** -2.5 expression pattern which is evidence for compartment-specific ***nkx*** -2.5 regulatory modules. A 505 bp regulatory element was identified that contains multiple GATA, NKE, bHLH, HMG and HOX consensus binding sites. This element is sufficient for gene activation in the ***cardiac*** crescent and in the ***heart*** outflow tract, pharynx and spleen when linked directly to lacZ or when positioned adjacent to the hsp68 promoter. Mutation of paired GATA sites within this element eliminates gene activation in the ***heart*** , pharynx and spleen primordia of transgenic embryos. The dependence of this ***nkx*** -2.5 regulatory element on GATA sites for gene activity is evidence for a GATA-dependent regulatory mechanism controlling ***nkx*** -2.5 gene expression. The presence of consensus binding sites for other developmentally important regulatory factors within the 505 bp distal element suggests that combinatorial interactions between multiple regulatory factors are responsible for the initial activation of ***nkx*** -2.5 in the ***cardiac*** , thyroid and spleen primordia.
RE CNT 62 THERE ARE 62 CITED REFERENCES AVAILABLE FOR THIS RECORD.
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 16 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE 3
AN 1998:296822 BIOSIS
DN PREV199800296822
TI The ***cardiac*** tissue-restricted homeobox protein ***Csx*** /Nkx2.5 physically associates with the zinc finger protein GATA4 and cooperatively activates atrial natriuretic factor gene expression.
AU Lee, Youngsook (1); Shioi, Tetsuo; Kasahara, Hideko; Jobe, Shawn M.; Wiese, Russell J.; Markham, Bruce E.; Izumo, Seigo
CS (1) Cardiovasc. Res. Cent., Room 5720, Med. Sci. Cent., Univ. Wisconsin Med. Sch., 1300 University Ave., Madison, WI 53706 USA
SO Molecular and Cellular Biology, (June, 1998) Vol. 18, No. 6, pp. 3120-3129. ISSN: 0270-7306.
DT Article
LA English
AB Specification and differentiation of the ***cardiac*** muscle lineage appear to require a combinatorial network of many factors. The ***cardiac*** muscle-restricted homeobox protein ***Csx*** /Nkx2.5 (***Csx***) is expressed in the precardiac mesoderm as well as the embryonic and adult ***heart*** . Targeted disruption of ***Csx*** causes embryonic lethality due to abnormal ***heart*** morphogenesis. The zinc finger transcription factor GATA4 is also expressed in the ***heart*** and has been shown to be essential for ***heart*** tube formation. GATA4 is known to activate many ***cardiac*** tissue-restricted genes. In this study, we tested whether ***Csx*** and GATA4 physically associate and cooperatively activate transcription of a target gene. Coimmunoprecipitation experiments demonstrate that ***Csx*** and GATA4 associate intracellularly. Interestingly, in vitro protein-protein interaction studies indicate that helix III of the homeodomain of ***Csx*** is required to interact with GATA4 and that the carboxy-terminal zinc finger of GATA4 is necessary to associate with ***Csx*** . Both regions are known to directly contact the cognate DNA

sequences. The promoter- ***enhancer*** region of the atrial natriuretic factor (ANF) contains several putative ***Csx*** binding sites and consensus GATA4 binding sites. Transient-transfection assay indicate that ***Csx*** can activate ANF reporter gene expression to the same extent that GATA4 does in a DNA binding site-dependent manner. Coexpression of ***Csx*** and GATA4 synergistically activates ANF reporter gene expression. Mutational analyses suggest that this synergy requires both factors to fully retain their transcriptional activities, including the cofactor binding activity. These results demonstrate the first example of homeoprotein and zinc finger protein interaction in vertebrates to cooperatively regulate target gene expression. Such synergistic interaction among tissue-restricted transcription factors may be an important mechanism to reinforce tissue-specific developmental pathways.

L14 ANSWER 17 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE 4
AN 1998 72827 BIOSIS
DN PREV199800072827
TI Autoregulation of human ***cardiac*** homeobox gene CSX1: Mediation by the ***enhancer*** element in the first intron.
AU Oka, Toru; Komuro, Issei (1); Shiojima, Ichiro; Hiroi, Yukio; Mizuno, Takehiko; Aikawa, Ryuichi; Akazawa, Hiroshi; Yamazaki, Tsutomu; Yazaki, Yoshio
CS (1) Dep. Med. III, Univ. Tokyo Sch. Med., 7-3-1 Hongo, Bunkyo-ku, Tokyo 113, Japan
SO Heart and Vessels, (1997) Vol. 0, No. SUPPL. 12, pp. 10-14. ISSN: 0910-8327.
DT Article
LA English
AB ***Csx*** / ***Nkx*** -2.5 is a murine homeobox gene expressed predominantly in cardiocytes and their progenitor cells. The highly lineage-restricted expression pattern of ***Csx*** / ***Nkx*** -2.5 gene suggests the existence of a positive autoregulatory loop in the transcriptional regulation of ***Csx*** / ***Nkx*** -2.5. The first intron of CSX1, a human homolog of ***Csx*** / ***Nkx*** -2.5 gene, had two potential CSX1-binding sequences. Activity of the CSX1 minimal promoter in cultured ***cardiac*** myocytes was significantly increased by placing the 3' half of the CSX1 first intron downstream of the reporter gene, suggesting that this region functions as a positive ***enhancer*** element. Transient transfection experiments in nonmuscle cells demonstrated that the reporter construct containing the CSX1 minimal promoter and the 3' half of the CSX1 first intron was strongly transactivated by overexpression of CSX1, whereas the CSX1 minimal promoter alone was not. Together these results suggest that the highly lineage-restricted expression of CSX1 is accomplished by autoactivation, which may be mediated by the ***enhancer*** element in the first intron.

L14 ANSWER 18 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 1997 100639 BIOSIS
DN PREV199799399842
TI Expression of ***cardiac*** muscle markers in rat myocyte cell lines.
AU Engelmann, Gary L. (1); Worrell, Robert A.; Duff, Richard A.; Grutkoski, Patricia S.; Chien, Kenneth R.; Harvey, Richard P.
CS (1) Dep. Med., CVI Room 5224, Building 110, Loyola Univ. Sch. Med., 2160 South First Ave., Maywood, IL 60153, USA
SO Lamers, J. M. J. [Editor]; Verdouw, P. D. [Editor]. (1996) pp. 87-91. Developments in Molecular and Cellular Biochemistry, 17; Biochemistry of signal transduction in myocardium. Publisher: Kluwer Academic Publishers, PO Box 989, 3300 AZ Dordrecht, Netherlands. Meeting Info.: Satellite Symposium of the 15th World Congress of the International Society for Heart Research, Rotterdam, Netherlands, June 30-July 1, 1995. ISBN: 0-7923-4067-1.
DT Book, Conference
LA English

L14 ANSWER 19 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE 5
AN 1996 282866 BIOSIS
DN PREV199699005222
TI Transcriptional regulation of a mouse Clara cell-specific protein (mCC10) gene by the ***NKx*** transcription factor family members thyroid transcription factor 1 and ***cardiac*** muscle-specific homeobox protein (***CSX***.
AU Ray, Manas K.; Chen, Ching-Yi; Schwartz, Robert J.; Demayo, Francesco J. (1)
CS (1) Dep. Cell Biology, Baylor Coll. Med., One Baylor Plaza, Houston, TX 77030, USA
SO Molecular and Cellular Biology, (1996) Vol. 16, No. 5, pp. 2056-2064. ISSN: 0270-7306.
DT Article
LA English
AB This report defines the elements between bp -800 and -166 that regulate the quantitative level of mouse CC10 (mCC10) transcription in the lungs. The elements in this promoter domain are the response elements for the ***NKx*** 2.1 homeobox protein, thyroid transcription factor 1 (TTF1). DNase I footprint analysis identified five binding sites for TTF1 between bp -800 and -166. These sites are located at bp -344 to -335, -282 to -273, -268 to -263, -258 to -249, and -199 to -190. In addition to these ***enhancer*** elements, two TTF1 binding sites were identified in the proximal promoter region (bp -166 to +1), at bp -74 to -69 and -49 to -39. An identical footprint of the mCC10 promoter region was also observed with another member of the ***NKx*** family, ***NKx*** 2.5, the ***cardiac*** muscle-specific homeobox protein (***CSX***). Deletion and linker-scanner mutational analyses of the TTF1 binding sites in the mCC10 distal promoter region with transient cotransfection into CV1 cells with either TTF1 or ***CSX*** identified the site located between bp -282 and -273 as the major regulator of CC10 expression, with minor regulation by sites at bp -344 to -335 and -258 to -249. The importance of the ***NKx*** binding site at bp -282 to -273 was verified in vivo. Transgenic mice generated with the human growth hormone gene fused to 800 bp of the mCC10 promoter containing a mutation in the TTF1 binding site at bp -282 to -273 showed a reduction in transgene expression equal to that of the mice generated with only 166 bp of 5'-flanking DNA. This report emphasizes the importance of TTF1 or related factors as major regulators of pulmonary gene expression and demonstrates the potential of ***NKx*** proteins to bind and activate heterologous target genes.

L14 ANSWER 20 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 1996 312919 BIOSIS
DN PREV199699035275
TI Transcription factors and the ***cardiac*** gene programme.
AU Doevendans, Pieter A.; Van Bilsen, Marc (1)
CS (1) Dep. Physiol., Cardiovasc. Res. Inst. Maastricht, Univ. Limburg, PO Box 616, 6200 MD, Maastricht, Netherlands.
SO International Journal of Biochemistry & Cell Biology, (1996) Vol. 28, No. 4, pp. 387-403. ISSN: 1357-2725.
DT General Review
LA English
AB During the past decade, major advances have been made in uncovering the mechanisms that switch genes on and off. Gene methylation and histones play an important role in gene (in)activation. Following gene activation, the initiation of transcription by RNA polymerase requires the assembly of multiple protein complexes on the promoter region of a gene. How a cell type-specific gene expression pattern can be induced is a key question in cardiovascular biology today. Members of the helix-loop-helix-family of the transcription factors play a dominant role in skeletal muscle formation. In ***cardiac*** muscle the situation is less obvious. Recent studies identified muscle transcription factors like MEF-2, TEF-1 and MNF, which are common to both the skeletal and ***cardiac*** muscle lineages. A few transcription factors, among which ***Nkx*** 2.5 and GATA-4, are expressed predominantly in the ***heart***. The absence of master regulators in the ***heart*** points to the importance of interaction between ubiquitous factors and tissue restricted factors to initiate the ***cardiac*** gene programme and to lock these cells in their differentiated state. The recent development of murine transgenic and gene-targeting technology provides tools to study the role of mammalian transcription factors in vivo. Interesting ***cardiac*** phenotypes are found in gene targeted mice, indicating a crucial role for retinoic acid and homeobox genes in murine cardiogenesis.

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TI Expression of ***cardiac*** muscle markers in rat myocyte cell lines
AU Engelmann, Gary L.; Worrell, Robert A.; Duff, Richard A.; Grutkoski, Patricia S.; Chien, Kenneth R.; Harvey, Richard P.
CS Cardiovascular Institute, Loyola University, Maywood, IL, 60153, USA
SO Mol. Cell. Biochem. (1996), 157(1&2), 87-91. CODEN: MCBIB8, ISSN: 0300-8177.
DT Journal
LA English
AB Recently developed rat ***heart*** myocyte cell lines have afforded us the opportunity to evaluate the expression of several transcription factors assocd. with early ***cardiac*** development. These factors include, but are not limited to, ***Nkx*** -2.5/ ***Csx*** , MEF-2C and MLP (Muscle LIM Protein). These factors have been shown to be temporally expressed in pre- ***cardiac*** mesenchyme coincident with the earlier stages of ***heart*** development. Using the BWEM and CLEM myocyte cell lines as models of the embryonic, committed cardiomyocyte, we have evaluated the basal expression levels of these three genes over multiple passages. Both cell lines express these genes, with MEF-2C being the most abundant based on Northern blot hybridization analyses. Interestingly, as these cells increased their passage no., there was a corresponding increase in their basal expression levels. To evaluate potential 'downstream' effectors of these genes, we examd. the basal expression levels of two ***cardiac*** -specific genes cTNC and MLC-2v. Transcript levels for both of these contractile filament genes were elevated with passage, suggestive of a inductive process mediated by one or all of these three transcription factors. Promoter anal. of MLC-2v expression in the CLEM line shows that this increase is transcriptionally-mediated and the lines retain the necessary regulatory factors to maintain and control the transcription of these genes. Anal. of the dynamics of the regulatory role(s) that these three transcription factors play in ***cardiac*** development can now be evaluated in a homogeneous, cell culture system.

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